Claims

1. A peptide having 2 to 10 amino acids or a derivative thereof which is able to restore wild type function of human p53 for use in therapy.

2. Use of a peptide having 2 to 10 amino acids or a derivative thereof which is able to restore wild type function of human p53 in the manufacture of a medicament for treating cancer.

3. A peptide, derivative or use as claimed in claim 1 or claim 2 having 3 to 7 amino acids.

4. A peptide, derivative or use as claimed in any one of claims 1 to 3 wherein said peptide incorporates the tri-peptide sequence WCT.

5. A peptide, derivative or use as claimed in claim 4 wherein said peptide incorporates the pentapeptide sequence M-G/M/V-WCT.

6. A peptide derivative or use as claimed in any preceding claim wherein said peptide derivative has been modified at the C and/or N terminus to include a signalling or targetting moiety.

7. A peptide derivative or use as claimed in claim 6 wherein said signalling or targetting moiety is selected from the group comprising folate and the HIV Tat translocation sequence.

8. A method of screening a library of molecules for the ability of members of that library to restore or modify the function of a target protein in an intra-cellular environment, which method comprises introducing the library into host cells which have a reporter system which allows the identification of those cells in which the function of the target protein has been restored or modified.

9. A method as claimed in claim 8 wherein the target protein is a nucleic acid binding protein.

10. A method as claimed in claim 9 wherein the nucleic acid binding protein is p53.

11. A method as claimed in any one of claims 8 to 10 wherein the reporter system comprises a reporter gene which is operably linked to a sequence of nucleotides which provides a binding site for the target protein or for a protein which associates with or is a substrate for said target protein.

12. A method as claimed in claim 11 wherein the reporter gene is operably linked to a p21 or Bax promoter.

13. A method as claimed in claim 11 or claim 12 wherein the protein product of the reporter gene includes a secretion signal peptide.

14. A method as claimed in any one of claims 11 to 13 wherein the protein product of the reporter gene includes a transmembrane domain.

15. A method as claimed in any one of claims 11 to 14 wherein the host cells have been transfected with said reporter gene.

16. A method as claimed in any one of claims 8 to 15 wherein the molecular library is a peptide library.

17. A method as claimed in claim 16 wherein said peptides have 2-8 amino acids.

18. A method as claimed in claim 16 or claim 17 wherein the library is introduced into the host cell population in the form of nucleic acid constructs which encode the peptide library.

19. A method as claimed in any one of claims 16 to 18 wherein each member of said peptide library has the sequence M-G/M/V-$(X)_n$, wherein n is an integer from 3 to 18, M is methionine, G is glycine, V is valine and each X, which may be the same or different, is any genetically coded amino acid.

20. A method as claimed in any one of claims 8 to 19 wherein the molecular library has at least 500 different members.

21. A method as claimed in any one of claims 8 to 19 wherein the host cells are eukaryotic cells.

22. Use of a compound identified by a method as claimed in any one of claims 8 to 21, or a derivative of such a compound, in restoring wild-type function to a mutant

protein.

23. Use of a compound identified by a method as claimed in any one of claims 8 to 21, or a derivative of such a compound, in the manufacture of a medicament for restoring wild-type function of a mutant protein.

24. Use of a compound identified by a method as claimed in any one of claims 8 to 21, or a derivative of such a compound, said compound being capable of restoring wild-type function of a mutant protein in the manufacture of a medicament for use in the treatment of a condition selected from cancer, cystic fibrosis, sickle cell anaemia, phenylketonuria, multiple carboxylase deficiency, methylpurine DNA glycosylase deficiency (MPG), ataxia and chemotherapy resistance due to mutations in the gene coding for methylguanine - DNA methyl transferase (MGMT).

25. Use of a compound identified by a method as claimed in any one of claims 8 to 21 in a design process to manufacture a pharmaceutical.

26. A method of treating cancer in a patient which method comprises administration of a peptide having 2 to 10 amino acids or a derivative thereof which is able to restore wild-type function of human p53.